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THE MECHANISM OF THE INTRA-OCULAR PRESSURE IN MAMMALIA

BY

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NOTTINGHAM

THE simplest procedure by which the nature of the intra-ocular pressure can be studied is by the ordinary method of ophthalmoscopic examination[1]. All that is necessary is to observe the most proximal portion of the central retinal vein at the disc, particularly if there is a physiological cupping, and note the constriction which takes place the instant one touches the eyelid with the lightest of light touches. The pressure of the merest touch on the skin, transmitted through the lid to the intra-ocular contents, is sufficient to cause a narrowing of the lumen of the proximal end of the retinal vein at the disc, which disappears the moment the finger is raised. It is an extremely pretty and instructive phenomenon and one which, incidentally, is much more conclusive than any experiment that human ingenuity can devise. It is, in short, the key to the understanding of the problem of the mechanism of the intra-ocular pressure.

The prompt response of the retinal veins to the slightest touch on the lids, proves how exquisitely balanced are the intra-ocular pressure and the venous pressure at the points of venous exit. Although only the exit of the central retinal veins is visible and

open to inspection, it is obvious that a similar pressure response takes place simultaneously at every other point of venous exit, including the venae vorticosae and Schlemm's canal. Incidentally Schlemm's canal is composed of merely a single layer of endothelium and thus considerably thinner than the retinal veins or venae vorticosae. By the use of our staple instrument, the ophthalmoscope, the question of the nature of the intra-ocular pressure and its equilibrium with the venous exit pressure in the eye is placed beyond discussion and above discordant laboratory findings. Further, the absence of any venous pressure response in accommodation or convergence demonstrates that, contrary to accepted views, action of the intra- or extra-ocular muscles produces no increase of intra-ocular pressure.

The extreme lightness of the touch which will produce the venous response gives an important indication of the level of the intra-ocular pressure. It supplies the requisite clinical proof that the intra-ocular pressure is in equipoise with the pressure within the veins as they pass out of the globe. This is exactly what Leonard Hill[2] found in the brain when he showed that the venous pressure in the torcular Herophili, a point of venous exit in the brain, is the same, a matter of 10 mm. Hg or 130 mm. H_2O, as the intra-cranial pressure. The two pressures always move together so that variations in the one always produce equal and corresponding changes in the other. The venous pressure response at the disc is a practical demonstration that the same level of pressure and the same mechanism exist in the eye as in the brain. Remembering the unity in Nature's workings, this is after all only what one would expect. When Nature has evolved a mechanism, she may and does alter the details, but the working principles concerned are always constant. The fact that the eye is a special sense organ is no adequate reason for endowing the tissues that enter into its structure with a special physiology at variance with the general principles that apply to similar tissues elsewhere in the body. The nerve elements subserving vision, the optic nerve and retina, constitute, embryologically, a lobe of the brain, yet at the lamina cribrosa, they are conceived as coming under physiological conditions differing materially, both in nature and level of pressure, from those existing in the brain.

The Experimental Intra-ocular Pressure

The practice of lumbar puncture has verified Leonard Hill's findings and the intra-cranial pressure is recognised as being about 10 mm. Hg or 130 mm. H_2O. Intra-ocular pressure as measured manometrically by numerous observers has been found to be in the neighbourhood of 25 mm. Hg or 330 mm. H_2O. This figure,

no less than two and a half times greater than the intra-cranial pressure, has long been generally accepted, but the circumstances responsible for such high readings, though visible and open to inspection by ophthalmoscopic examination, have never been taken into account. No thought has been given to what happens to the circulation inside the eye when a not too sharp canula is driven into the anterior chamber.

The effect of the localised application of pressure on the globe is similar to that produced in the hydraulic press, every square millimetre of the internal surface of the globe is subjected to the same pressure, sufficient to check the circulation and exsanguinate the eye temporarily. As soon as the canula has entered the anterior chamber, the circulation is restored with a bound. The action and reaction is felt by every cell in the eye, tissue pressure is affected, secretory and circulatory pressure raised. Under such conditions it is only to be expected that the manometric findings average 25 mm. Hg. Such readings are, however, but the reaction of the intra-ocular contents and circulation to the sudden pressure they have been subjected to. If one were to waylay a man, slap him on the face and then forcibly take his blood pressure, one would hardly expect such readings to be exactly physiological, regardless of the number of individuals on whom this experiment was made. Similarly the experimental finding of an intra-ocular pressure of 25 mm. Hg is the expression of the eye's reaction to the assault it has sustained. One admits the manometric findings but the conditions under which these high figures have been obtained must be taken into consideration.

If the experimental figure of 25 mm. Hg is to be accepted as the physiological level of the intra-ocular pressure, then, if correct, there should be no difficulty in co-ordinating it with other relevant conditions. After all correlation is fundamental to the correct interpretation and understanding of all physiological processes, but as soon as one attempts to do so in the eye, one encounters all sorts of involved and anomalous situations whose existence has been overlooked.

Problems arising from the alleged High Physiological Level of the Intra-ocular Pressure

The first problem is the very natural one, why the eye should require such a high level of pressure. This, it is explained, is necessary to keep the eyeball distended and maintain its form as an optical instrument. In other words the globe is not rigid enough, but like a toy balloon, it must be kept inflated to maintain a definite shape. A study of the corneo-sclera in mammalia shows that this fibrous envelope is, however, more than sufficiently thick

to maintain the shape of the eye, without the necessity of a high intra-ocular pressure. The maintenance of the form of the mammalian eye depends not on the pressure exerted from within, but on the rigidity of its covering. We have in our daily work, ample evidence of this when performing an intra-ocular operation. If the aqueous exerted a pressure equal to 13 inches of water on the inner surface of the cornea, it would be impossible to make one's incision for extraction of cataract without fluid squirting out instead of merely oozing away, while at the same time the globe, being released from its internal pressure, should collapse.

The conditions at the disc present a whole series of problems which are quite incompatible with a high level of intra-ocular pressure (Fig. 1). To begin with the lamina cribrosa interposed between the intra-ocular and intra-cranial components of vision, is the weakest area of the corneo-scleral envelope. As its name implies, its construction is eminently unfitted to withstand any

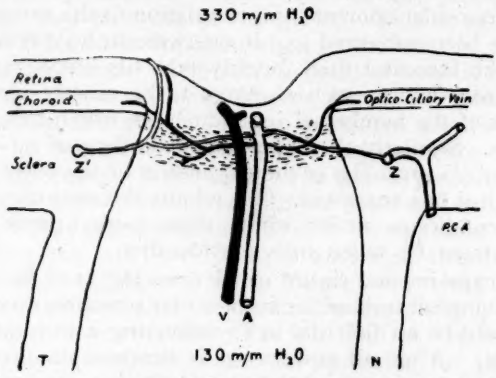

FIG. 1. Diagrammatic Section of the Optic Nerve Head.

The lamina cribrosa marks the anatomical boundary between the intra-ocular contents with alleged pressure of 330 mm. H_2O and the optic nerve with pressure of 130 mm. H_2O. Central retinal artery (A) and vein (V). On nasal side (N), the posterior ciliary artery (PCA) gives a branch to form the arterial circle of Zinn (Z), which anastomoses with the central retinal artery. On the temporal side (T) the circle of Zinn (Z') gives off the cilio-retinal artery. The cilio-retinal vein joins the central retinal vein (V). On the nasal side an optico-ciliary vein from the choroid drains into the retinal vein.

pressure, yet such is taken for granted, but does not make it any more possible.

The question of the venous return at the disc is even more peculiar. The anastomosing branches of the arterial circle of Zinn (Fig. 1, Z) are associated with a corresponding capillary venous network which joins the central retinal vein and a passing thought

should be spared as to how they carry on between the upper millstone of the intra-ocular pressure and the nether of the intra-neural.

Similarly the unobtrusive existence in the lamina cribrosa of the optico-ciliary veins (Fig. 1) that pass from the choroid to the central retinal vein or one of its ramifications, is somewhat of an enigma. Fluids always flow in the direction of least resistance, yet the choroidal venous return though linked with the central retinal vein, makes no use of this route to short-circuit the venae vorticosae and escape out of the high intra-ocular pressure. One can only ask why and obtain an unresponsive echo.

From these unconsidered trifles one must pass to the consideration of the central retinal vein itself (Fig. 1, V). The pressure phenomenon already detailed, shows how responsive the venous wall is to the very slightest variations in the intra-ocular pressure. If, in the globe, the venous wall is subjected to a pressure of 330 mm. H_2O, what happens when the vein has passed through the lamina cribrosa and lies in the optic nerve with a tissue pressure of only 130 mm. H_2O? There is no anatomical evidence that the vein distends or that its walls are in any way altered to meet the internal pressure they are now suddenly called upon to withstand. Veins are not in the habit of withstanding long continued high pressure, yet that is exactly what the central vein in its course through the optic nerve is expected to do. Incidentally too, the same question arises in the case of the venae vorticosae, for having passed through their oblique course in the sclera, how come these veins to resist such high internal pressure without support? The problem has only to be stated to stand out as contrary to all known physiological conditions.

From the clinical aspect the alleged physiological disparity of pressure on either side of the lamina cribrosa shows up even more incongruously in papilloedema. The manifestations of venous engorgement and oedema present at the disc in this condition, are the direct product of a raised intra-cranial pressure[3]. To produce either venous congestion or oedema it is necessary that the intra-cranial pressure rise above the intra-ocular, *i.e.*, above 330 mm. H_2O.

It is an established clinical fact from lumbar puncture, that an intra-cranial pressure approaching 330 mm. H_2O is a very high one, yet marked papilloedema is present with intra-cranial pressure very much lower than the supposed physiological intra-ocular pressure. How can oedema and venous engorgement appear at the disc when the pressure on the cranial side of the lamina is still below that of the ocular? (Fig. 1). Papilloedema should be a physical impossibility except in those cases where the intra-cranial pressure has risen above 330 mm. H_2O. One has not exhausted

the list of similar paradoxes, but will conclude with a final clinical anachronism.

Thanks to the pioneer work of Lagrange, every surgeon has produced " fistulisation " of the corneo-sclera with a visible opening between the anterior chamber and the sub-conjunctival tissue. The resulting " filtering cicatrix " raises the question of how the sub-conjunctival tissue can support the intra-ocular pressure. Tonometric readings, for what they are worth, may indicate " subnormal tension," but then under such conditions, how comes the eye to function as an optical instrument? A high physiological level of pressure is regarded as essential to keep the globe a rigid optical apparatus, but on the other hand, an eye that has been successfully trephined demonstrates that such is not at all necessary. One cannot expect to have it both ways, and furthermore Nature is not in the habit of working at higher levels than absolutely necessary. This must be particularly the case when conformity with associated physiological conditions cannot be established.

The Physiological Equilibrium between the Intra-ocular and Intra-cranial Pressures

If the above objections are valid, then I submit that my published experimental findings with Leonard Hill[4] are not only more likely to be correct than those generally accepted, but in addition, as they are supported by clinical proof, that they present a complete and simple explanation of the mechanism of the intra-ocular pressure in mammalia.

We found that simultaneous measurement not only gave the same level of pressure as in the torcular Herophili, about 10 mm. Hg, in both brain and eye, but these pressures rose and fell together under all circumstances. This physiological conformity is after all only what one would expect from a recognition of the following conditions:

(1) Fluids are incompressible, transmit pressure equally in all directions and invariably lie at the lowest attainable hydrostatic level.

(2) Within the closed coverings of the brain and eye, the blood stream flows through an elastic, and not a rigid, system of tubes, and the pressure within the veins as they pass out of the spheres, is the lowest circulatory pressure.

(3) In the eye, as in the cranium, the fluids lie at venous exit level of pressure because this is the lowest circulatory pressure; hence the retinal venous pressure phenomenon at the disc.

Neither the brain nor the eye possesses any extraneous mechanism to protect them from changes in the general venous or general

arterial pressures. A rise in vena cava pressure makes itself felt at once by backward pressure and the pressure within the brain and eye rises millimetre for millimetre with that of the vena cava. On the other hand a rise of arterial pressure in the aorta only produces a proportional rise of pressure, as between the aorta and venous exits in brain and eye, lie the unknown and varying resistance of the arterioles.

In other words the intra-cranial and intra-ocular pressures, while varying with venous exit pressure and influenced directly by that in the vena cava, can, on the arterial side, be regarded as representing the pumping force of the heart left over after the blood has been driven through and has overcome the resistance of the arterioles.

Leonard Hill had to lay stress on the rigid nature of the cranium to emphasise that it was circulatory pressure and not volume that is responsible for the intra-cranial pressure. The principle of the circulatory nature of the intra-cranial pressure having been once established, the nature of the covering is immaterial so long as the contents are physiologically enclosed, and this applies equally to the infantile cranium with its open fontanelles and variable volume, as to the eyeball. The material fact is that it is the general circulatory pressure on the venous side which governs the physiological level of the intra-cranial and intra-ocular pressures and maintains them in the neighbourhood of 130 mm. H_2O. With such a mechanism and equilibrium the problems enumerated above cease to exist.

The Intra-ocular Tension

If the manifestation of the venous pressure response is carried a stage further and the disc kept under observation while the globe is palpated with the finger of the disengaged hand, the sequence of events which give rise to the appreciation of the intra-ocular tension can be followed.

As the fingers are placed in position one will first perceive the immediate constriction of the retinal termination of the venous exit. As the globe is palpated one will note, most readily in children, that a retinal arterial " pressure pulse " is produced, similar to that in glaucoma. This pressure or collapsing pulse is created in consequence of the applied pressure being greater than the diastolic pressure, so that only the systolic arterial wave can pass into the eye.

This clinical observation shows that the feeling of tension imparted to the fingers, when palpating the globe, is actually due to the pressure which the fingers are exerting on the eye. The intra-ocular pressure is thereby raised temporarily from the low

level of venous exit pressure, manifested by venous pressure response, to the higher level of diastolic arterial pressure, as shown by the collapsing arterial pulse.

The intra-ocular tension as tested clinically, is nothing more than the feeling of the range or depth of resilience of the globe, when, by palpation, its internal pressure is raised from its normal venous exit level towards the arterial side of the intra-ocular circulation. In glaucoma the intra-ocular and venous exit pressures being correspondingly high the range of resilience of the globe is proportionally less and in consequence the eye feels hard.

Summary

(1) A series of problems, clinical and physiological, are enumerated which are inexplicable, if as alleged the intra-ocular pressure is two and a half times greater than the intra-cranial pressure. They cease to exist with the realisation that the same mechanism and the same level of pressure, about 10 mm. Hg, obtain in the eye as in the brain.

(2) The key to the understanding of the mechanism of the intra-ocular pressure is the reaction of the central retinal vein at the disc to the slightest touch on the lid, supplying clinical proof of the equilibrium between the intra-ocular pressure and venous exit pressure.

(3) The intra-ocular pressure is not dependent on volume but, as in the brain, it is maintained and varies with venous exit pressure because this is the lowest circulatory pressure in the closed sphere of the globe.

(4) It is the general circulatory pressure on the venous side which governs the physiological level of the intra-cranial and intra-ocular pressures and maintains them at 10 mm. Hg. Every variation in vena cava pressure is reflected backward millimetre for millimetre in both brain and eye. Variations in arterial pressure only produce a proportional change, as between the aorta and the venous exits in brain and eye the unknown and varying resistance of the arterioles has to be overcome.

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DISTANCE OBJECT TEST TYPE FOR CHILDREN

BY

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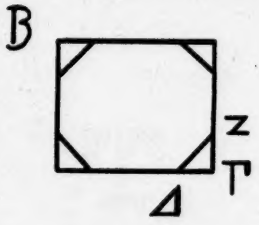
WHEN preparing this children's chart last winter I noticed that if the chart were placed sufficiently away from me the square on the 6/6 line lost its angles and appeared to be more or less round. I was not conscious then of the fact that the Greek mathematical friend of our youth, Euclid, 300 B.C., was familiar with this and as a matter of fact in his observations regarding it we have the first visual test on record. A translation of what he wrote is as follows:—

Rectangular magnitudes when seen from a distance become peripheries (or curves). Let the right angled figure *BΓ* be elevated and seen from a distance. Then since each visible thing has a



certain measure of distance at which it can no longer be seen, the angle *Γ* does not appear, and only the points *ΔZ* are seen. This will happen equally in the case of the three remaining angles. Thus the whole will appear to be a periphery (or curve).

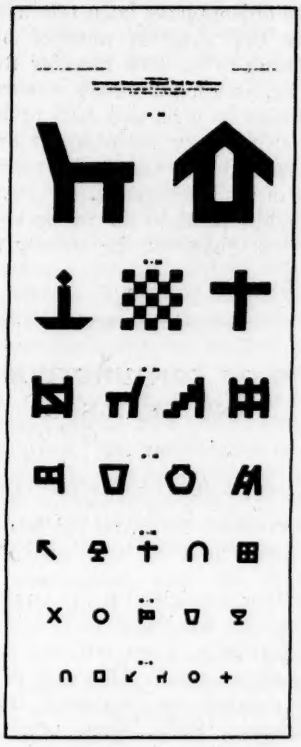
Except for the mentioning of mustard seeds and other tiny seeds being used in testing visual acuity, history gives us little further information until the time of that famous English experimental philosopher, Robert Hooke (1635-1703). His optical investigations led him to adopt in an imperfect form the undulatory theory of light and he made independent observations on diffraction. However, it was his interest in the heavenly bodies that brought to his notice that two of these must be separated by nearly 60 seconds if they are to be recognised by the naked eye and it was this observation which has been the basis of practically all test charts since his day. Though 60 seconds has been accepted as the angle of visual acuity yet many people can see images separated when the angle is as small as 24 seconds. If we assume that the posterior nodal point is 15·6 mm. from the retina, then an angle of 24 seconds corresponds with a distance between the images at the retina of

·0018 mm. The diameter of a cone at the macula is about ·002 mm. so that there is an interesting relationship between the limit of perception and the anatomical structure of the retina.

It was Snellen in 1862 who first prepared letters, the component parts of which subtend an angle of 1 minute at 6 metres while the whole letter subtends an angle of 5 minutes. Although we often speak of this test made at 6 metres as being an infinity test, yet at this distance we actually use one prism dioptre of convergence and 0·16 dioptre of accommodation. An infinity test would, of course, have to be at infinity but I think if test charts were placed 8 metres away, we might regard the amount of convergence and accommodation at that distance as being negligible. Since most of us test at 6 metres we ought to bear these facts in mind when some of our patients complain that they can see aeroplanes better in the sky when they remove their distance glasses. Notwithstanding these imperfections the Snellen test has by its popularity and its continued use shown itself to be of great practical value.

A glance at the accompanying diagram will demonstrate the one and five principle, as it is sometimes called, on which a Snellen letter is constructed. I have endeavoured to produce a children's chart with drawings of familiar objects built up on the same

principle. The necessity for such a chart came to me when I realised that we cannot expect a child to see comfortably the print in the present day school books if his visual acuity for distance is not 6/12 or better. What then can we say about the child who cannot yet read but is doing handicraft in his kindergarten class which requires just as much visual acuity? How can we be certain that he can get 6/12 or better? The familiar children's chart with horse, dog, cat, etc. can only give us a vague idea of a child's vision. How vague that idea is can be realised if we compare the chart with a Snellen's by means of a No. 1 Evans occluding glass. The former chart, however, makes no pretence of accuracy and there is no indication of the value of its objects. Unfortunately, some children's charts, with no more claim to precision, do actually bear Snellen $\frac{D}{d}$ values and are positively misleading. At present there are only a few children's charts on the market which accurately conform to the Snellen principle. Amongst these might be mentioned the E chart introduced as far back as 1885 by Albini and in later times by Bishop Harman, and the "broken-ring"



chart, which was the work of Landolt and was popularised by the Egyptian Government. As a matter of fact these charts were mainly intended for illiterates, and oculists who make use of them can hardly expect them to be interesting to children or easy to use with them.

The trial chart I had printed contained 32 test objects and was tried out at school clinics in different parts of the country. In the

present one only those objects have been retained which were most easily interpreted by the greatest number of children. Some objects which might seem to be very familiar to adults had to be rejected. For instance, an anchor which was easily recognised by boys presented difficulties to girls and had to be discarded. The objects retained are either easy to interpret or else capable of a varied interpretation so that the test is an estimate of the child's visual acuity and not of his intelligence.

Though the work is independent on my part yet I now find that the idea of an object test chart on the strictly Snellen principle is by no means original.

ARGYROSIS OF CONJUNCTIVA, CORNEA AND TEAR-SAC

BY

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THE deposition of silver particles on the conjunctiva is not at all rare. In countries where trachoma is endemic the use of silver drops is practised regularly, even without medical prescription, because of the relief experienced. One case is reported of a Czech farmer's wife who carried on treatment for twenty-two years (Knapová and Bruckner, 1922). Many oculists will be surprised at the length of time certain patients are using their prescriptions, the following being an example. A woman, aged 72 years, with cataract, incipient in one and more advanced on the other eye, was given argyrol ointment to be used for four days prior to operation. When she returned a year later she had a marked argyrosis, and it was discovered that she had used the ointment daily for the whole period. Successful intracapsular extraction failed to reveal any silver in the lens capsule.

Argyrosis of the conjunctiva arises not only after local use of silver, but also when this is administered orally and intravenously (Salus, 1917, Ascher, 1924), or in industrial diseases (Subal, Freyler, 1936), following treatment of nasal sinuses and after the use of silver to dye the beard, etc.

While argyrosis of the conjunctiva is frequent, corneal argyrosis is rarely mentioned (Ascher, Knapová and Bruckner, Vogt, Freyler, Kotsuka). My impression is that corneal argyrosis has been overlooked because it is not clinically conspicuous.

There are some cases of argyrotic tear-sac already described (Gabrielides, 1934; Damel, 1934; Ciotola, 1937). Undoubtedly such changes of the tear-sac would be discovered more frequently in cases of conjunctival argyrosis if it were possible to examine the sac histologically.

A case which I followed clinically provided me with anatomical material from the conjunctiva and the tear-sac, and presents some interesting questions.

Mrs. H., aged 67 years, who complained of watering of the right eye for some years was treated with dark drops which she used twice daily for some months. Silver deposits were not visible except in the conjunctiva. Ectropion of the nasal part of the lower lid was present, and the inferior punctum was everted. On squeezing there was mucous discharge but no pus. The whole conjunctiva of the lower lid, the nasal part of the upper lid, and the entire bulbar conjunctiva, the caruncle included, was stained partly lead grey, partly chocolate brown. The bulbar conjunctiva was diffusely stained a bluish grey, the conjunctiva of the lids was brown and patchy. The caruncle was covered with minute cysts containing clear fluid, on its surface there was greyish-brown dust. With the small beam of the slit-lamp we could localise the dust below the epithelium. A drop of 2 per cent. dionine produced oedema and the pigment was raised with the epithelium. A minute greyish-brown spider-shaped network was situated in the oedematous fluid between the sclera and the epithelium. All these changes were more marked on the nasal than on the temporal side. No lymph vessels were visible either accompanying the blood vessels, or separate; and there was no dust surrounding the blood vessels suggesting the existence of lymph vessels (as Koeppe described in his text-book).

Macroscopically both corneae appeared normal. The left showed, with the slit-lamp, a tiny central cystic macula and normal corneal reflexes. With the narrow beam the *right* cornea gave a more intensive corneal beam appearing bluish-white in its posterior part. The region of Descemet's area was clearer shining than the anterior part of the cornea, this being more conspicuous in the lower half, but present in the upper corneal tissue as well.

With the broad beam Descemet's area showed rainbow colours with coarse grain, but without any special pattern. The deeper parts of the eye, lens, fundi, etc., were normal. V.A. R.E., 6/6. L.E., 6/12.

November 3, 1939. R.E. Ovoid excision of conjunctiva and superficial tarsal plate of 5 mm. extent. Three stitches were inserted, desired effect being obtained.

January 20, 1940. R.E. Sac excised. Watering now greatly reduced.

Anatomically the conjunctiva showed a diffuse sub-epithelial argyrosis leaving the *epithelium* entirely *free of silver particles*. These particles were not even present in a *narrow subepithelial* zone of varying diameter (Fig. 1). Most of the particles were distributed in long rows of fine droplets, some thicker than others, some in dark clusters with a diameter of 20-50 μ, and with an appearance similar to a brown jellyfish with its tentacles. The silver infiltration was not equal in all places, at some areas 40-50 μ, at others 150-170 μ deep. With the highest power (x10 eyepiece and oil immersion) we see more of the structure of these fibres. They may be in double contour (Fig. 2). The coarser droplets

FIG. 1.

Granulated short coffee-brown fibres.

FIG. 2.

Tiny Ag. particles within fibrocytes. 10/40. High power.

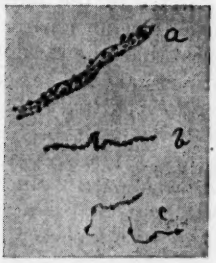


FIG. 3.

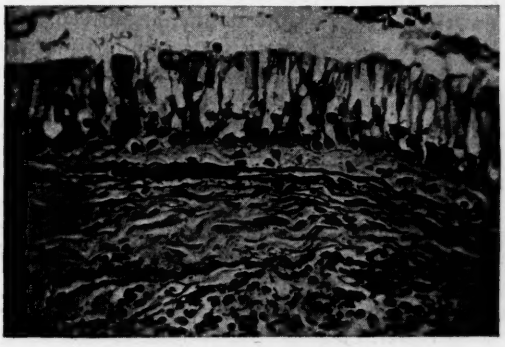
Conjunctival argyrosis. Three kinds of argyrotic fibres. 10/40

Selective argyrosis of elastic tissue.

FIG. 4.

Argyrosis of the tear-sac (Haem.-Eosin). Low power.

Goblet cells of sac-epithelium.



←silverfree subepithelial zone
←elastic elements

FIG. 5.

Argyrosis of the tear-sac (Haem.-Eosin). High power.

were restricted to the surface of the fibres (*a* in Fig. 3). Some fibres were thin, the particles lying narrow, close to each other (*b*). The tiniest were so thin that they were hardly visible, being marked by some particles at various distances (*c*). Besides the silver covered fibres *free particles* were found, mostly within the deep layers. Endothelial cells of the capillary system contained very few silver grains. The sac had a narrow empty lumen (Fig. 4), its epithelium was partly mucous degenerated, partly thickened to 10-12 rows, partly missing completely. *Nowhere was there a trace of silver within the epithelium.* Here as in the conjunctiva a narrow silverfree space was found (Fig. 5). The

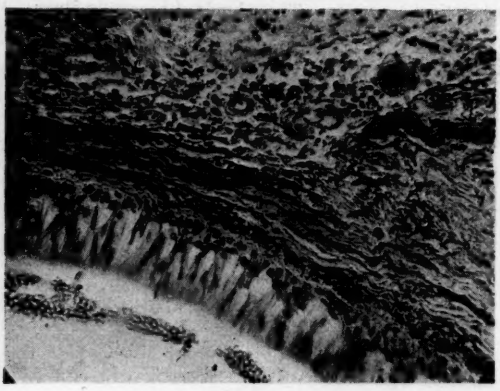


FIG. 6.

a. Selective staining of elastic fibres.
b. Brown granulated precipitates.
c. Shorter broken, punctate Ag. threads.

silver fibres underneath were richer than those of the conjunctiva, they ran mostly parallel to the epithelial surface, unequally distributed, but everywhere present in the slide round the lumen of the sac. They reached deeper than the conjunctival ones on the average 120 μ. Those close to the epithelium were of a brownish hue, the deeper ones were black. The elastic " ribbons " were thicker and longer than the conjunctival ones (Figs. 5, 6). There were more free particles. Some of them were thicker and aggregated to patches. Groups of thick patches black as china ink were packed subepithelially at certain spots. Endothelial cells of capillaries and precapillaries were partially filled with silver grains (Fig. 7). Some haemorrhages contained them too. Groups of large mast cells, some of them with a diameter of 12-17 μ (Figs. 8, 9), filled with equal, clear purple corpuscles and a light eosin-red

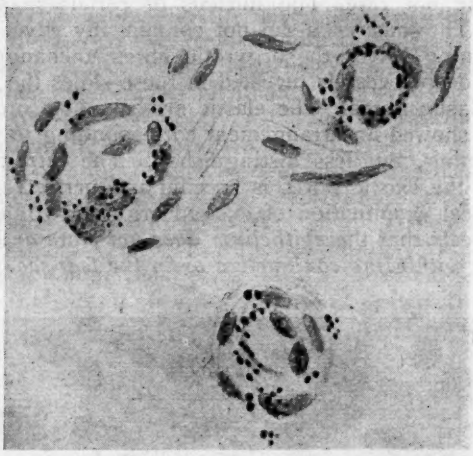


FIG. 7.

Endothelial phagocytosis—Argyrosis of tear-sac.

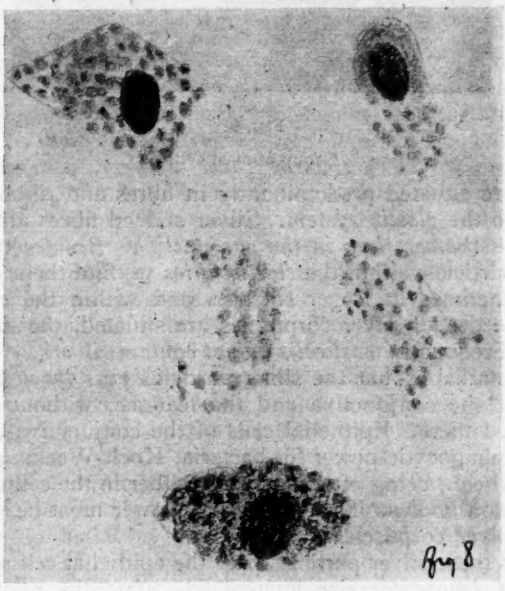


FIG. 8.

Mast cell groups in argyrotic sac.

plasma were not rare. The nucleus of certain mast cells had vanished. The mast cells did not contain any silver corpuscles. Orcein staining leaves the argyrotic fibres unchanged. Cross sections of deeper vessels revealed in these slides the presence of silverfree elastic fibres. The elastic system of an orcein stained control sac showed an arrangement corresponding to the " intra-vital " staining, but less distinguished. The *intra-vitam* silver staining of the elastic fibres is specific and perfect.

Histological examination of excised argyrotic conjunctiva and tear-sac reveals that the *epithelium does not store any silver particles either within the conjunctiva or in the tear-sac.* The silver

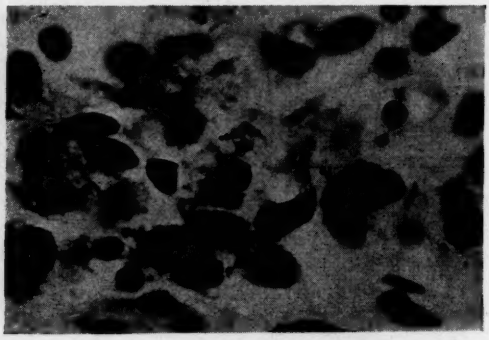


FIG. 9.

Group of mast cells.

particles are situated predominantly in fibres and ribbons corresponding to the *elastic system.* Silver stained fibres are *far more frequent in the sac than in the conjunctiva. Besides* the storage of silver particles within the *elastic fibres* we find them *free in the tissue,* sometimes in larger *clusters,* and within the *endothelial cells.* The deeper silver corpuscles are situated, the darker they appear. Frequently *mast cells do not contain silver.*

It is remarkable that the silver particles *pass through the epithelium* of the conjunctiva and the tear-sac, without being ad- or absorbed there. Epithelial cells of the conjunctiva are known to have a phagocytic power for bacteria, Koch-Weeks, gonococci, diplobacilli etc., being often packed together in the cellular plasma like inclusion bodies. This phagocytic power must be inconsiderable for silver corpuscles.

How do these silver particles pass the epithelial cell rows? We know that *leucocytes* rise immediately after the application of silver nitrate (unpublished own experiments). It may be that the silver particles are absorbed by the leucocytes which move actively

through the epithelium into the subepithelial tissue, where they perish and liberate what they have engulfed.

The greatest part of the silver particles are bound to the *elastic fibres*; a much smaller part is situated *free* within the connective tissue; the rest are " phagocytised " by the reticulo-endothelial cells of precapillaries and capillaries.

The above mentioned selective staining of the elastic elements may be interpreted either as an *adsorption*, or as *a chemical reaction*. Purely physical adsorption at the surface of the fibres would correspond very well with our findings, especially with respect to the double lined thick fibres. Nevertheless I am more inclined to the second view, as an adsorption which functions so selectively towards *one* kind of tissue does not appear very probable.

Silver nitrate precipitates halogens (Cl. Br, I) and sulphur. My conclusion was that Ag is precipitated as AgCl. and possibly reduced under the influence of light to metallic silver.

The other possibility seems to be that S containing cystin or cystein or one of its decomposition products reacts with the silver nitrate to form insoluble Ag_2S, a black precipitate.

These suppositions are founded on interesting experiments of (W. Heubner, 1937) who implanted 0·1 to 0·2 mm. silver platelets into corneal pockets of rabbits. These platelets were surrounded after some time by a tender connective tissue. Extirpation of the platelets with their sheaths and microchemical examination led to the discovery of *black crystals* which gave the reactions of silver sulphide. In between these there were found less frequently greyish white clusters of crystals which were proved chemically to be silver chloride. Both types of crystals look on simple microscopic examination like metallic silver. Heubner suggests that glutathione, a derivative of cystin, is present and represents the source of sulphur. It is easier to explain the effect of chlorides. On the other hand I could not trace an analytic investigation which would confirm the conclusion that elastic elements are *richer* in either *sulphur* or *chloride* than other tissues.

The following experiment was then carried out. A blenorrhoeic tear-sac was halved and the two halves brought into a 1 per cent. solution of silver nitrate. One of the halves was during this process exposed to light, the other one kept in the dark. Both pieces were then washed, embedded in paraffin and stained with haematoxylin and eosin. Most of the silver was found to be precipitated within the epithelial layer in large patches. In addition we also find many cells, both plasma cells and polynuclear leucocytes, covered with minute particles. The cellular boundaries are frequently defined sharply by thin lines of silver. There is *no selective adsorption on elastic fibres* and *no difference between the exposed and the dark portion.*

In the case under discussion it is rather amazing that silver reached the tear-sac, although it was choked. When the sac was rinsed all the liquid regurgitated mixed with tough mucus. It was assumed that no aspiration from the conjunctival sac could take place in a choked and obliterated sac or duct. This obviously has been proved to be incorrect. A very tiny aperture may still have remained unclosed and thus a feeble aspirating function of the canaliculi preserved. As soon as the silver particles would reach the sac, they could be deposited in the wall owing to the stagnation of the sac contents. On the other hand even if the sac is completely separated from its duct, a canaliculus may suck up the silver solution during lid closure. The sac then may deliver its surplus during the next winking of the eye.

The deep greyish blue reflecting corneal zone with its coarse " grain " belongs to the region of Descemet's membrane. Knapová and Bruckner suggest in accordance with Vogt, that this appearance is due to the silvery impregnation of the tissue *in front* of Descemet's membrane, which is especially rich in elastic fibres (Seefelder's " true lamina elastica corneae "). No decision on this point is possible without anatomical examination of a case. My own histological experience with rupture Descemeti and lipoidosis bulbi (to be dealt with separately) has convinced me that the true elastic lamina of the cornea is actually a thin anterior lamina *of* Descemet's membrane.

It is remarkable that endothelial cells of the sac arterioles absorbed silver particles, while on the other hand large mast cells did not contain any. Plasma cells are not infrequently found in cases of blenorrhoea of the tear-sac. Of mast cells I could find no previous observation.

The " elastophilia " of silver as observed in this case offers new prospects of experimental work. As *e.g.*, Ester Groenblad's syndrome (angioid streaks with pseudoxanthoma elasticum) has been proved to be an affection of the whole of the elastic system it would perhaps be useful to use silver compounds for the study of damage to the elastic system. We might even be able to *blockade the whole of the elastic system* by repeated intravenous application of silver compounds and thus eliminate its functions. Many other questions concerning the nourishment of ocular tissues and their metabolism may find their solution by such investigations.

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RETINOPATHIA CENTRALIS ANGIOSPASTICA (ANGIONEUROTICA), AND SEROSA ALLERGICA, AND THEIR RELATION TO DETACHMENT OF THE RETINA

BY

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CIRCUMSCRIBED disturbances of the retina, ophthalmoscopically recognisable, are found almost exclusively in the area of the macula, since a slight disturbance of function is not striking enough when a peripheral sector is concerned. A considerable number of such exudative processes in the macular area have been published as more or less rare solitary findings under different names. (Albrecht v. Graefe, Hirschberg, E. Fuchs, R. Batten, Kraupa, Asayama, Kitahara, Walsh and Sloan, Masuda, Oguchi, Guist, Reh, Hery, Junius, Stern, Suganuma, Kiewe and Reh, Cattaneo, Dollar, Abe, Bailliart, G. von Grolman, E. B. Streiff, Bangerta-Blaser, Avizonis. It is greatly to Horniker's credit (1929 and 1937) that he stressed the striking fact that the macular tissue is very easily injured by *vessel* disturbances. In several papers he described exactly the picture of retinitis centralis angioneurotica. Not infrequently narrowing of the capillaries of the retina, proved endoscopically, is the source of the affection. The present author himself has an experience of the astonishing figure of 72 cases, according to his last paper referring to this subject. They show a pathological form of reaction possibly of hereditary origin—an actual constitutional peculiarity. The angioneurotic constitution may have some connection with lues in the second and third generation (Kraupa), a question about which we are not yet well informed. The analogy to intermittent claudication, to migraine, to amaurosis partialis fugax and to bronchial asthma seems to be clear. Horniker measured the capillary pressure endoscopically with the ophthalmo-dynamometer. The visible rotating movement within the retinal blood vessels becomes slower under a certain pressure.

Kitahara (1936) on the other side describes a large series of cases of chorio-retinitis centralis in Japan. These he believes to be *tuberculous,* as many of the cases showed a picture like solitary tubercle. Horniker refutes any resemblance to a central solitary tubercle, and I agree with him. A positive tuberculin-prick-reaction alone does not entitle us to call an eye infection a tuberculous one. On the other hand I should like to emphasise at the beginning of these arguments that we must not always think of a caseation process when we speak of a tuberculous reaction of the tissue. It is much commoner for the tuberculous infection to occur histologically as an uncharacteristic, chronic, inflammation with or without production of an exudate—very often, too, without epithelioid or giant cells. Previously Allen K. Krause (1927) stressed the fact that we should not limit the use of the term " tubercular " to *nodular* formations, or we would overlook some of the commonest and most important anatomical manifestations of tuberculosis. Diffuse inflammation and suppuration may be caused by tubercle bacilli, these having little or nothing to do with the nodular structure. The central process in the retina might be tuberculous in its nature, even without a caseating tuberculous reaction, and different in its clinical picture. Streiff (1939) tries to separate chorio retinitis centralis serosa from Koniker's angiospastic retinopathy entirely.

Sanford Gifford and Marquart (1939) have communicated a series of eight cases lately, and emphasise the capillaro-spastic origin of the retinal changes. The affection is a transient one, and almost always disappears, with complete restoration of vision. As the changes in the ophthalmoscopic picture are very slight, the production of oedema cannot be anything else than the result of angiospasm. The authors found that their patients usually suffered from angiospastic changes in different parts of the body, that vaso-dilating drugs acted favourably, and neither tuberculosis, lues nor any blood disease was to be found. They summarised not only their own cases, but also all previous cases described as angiospastic, and used the non-committal term " retinopathy " in preference to " retinitis."

There seems to be a difference of opinion, not easily overcome. The one group (Kitasato, Riehm, Guist, etc.) call the central retinal process a specific one (in the majority tuberculous), the exudate being *infective* and *inflammatory*. Horniker, Gifford and others take the *vaso-spastic* character of the retinal change for granted, based on a constitutional state, and do not accept the idea of infection. It seems to be very difficult to bridge the gap between these two very different opinions.

I regret that my cases were seen some time ago, and that I have no contact with them now, so I cannot investigate the capillary

system as I had hoped to do, but there are a number of questions which still remain to be discussed, and new conditions and relations appear in connection with the description of new cases belonging to the very interesting group of transient macular oedema.

CASE 1.—K.R., aged 41 years, neurasthenic man; smokes five cigars daily; no alcohol; headaches very often, but no actual migraine; no tuberculosis, no lues, no anomalies of the vessels or spastic changes; nasal sinuses normal; teeth (X-ray plate) normal; complains of seeing colours, and has micropsia and metamorphopsia. All objects appear at first bright red, then yellow, green and finally violet. At the point of fixation he sees a dark spot. Left eye normal—vision 6/5.

Right Eye. No exophthalmos; normal eye movements; no pain on retropulsion; anterior part of the eye normal; media clear; disc normal; no papilloedema. In the macular region opaque and slightly elevated area without sharp limits. Relative central scotoma for all colours with 3 mm. objects. All colours are opaque, vague and undefined. The angle of the central scotoma is 6-8°. Vision 6/18 without correction (a year previously when controlled by me 6/5). Treatment: Sweating cure, old tuberculin intracutaneously (always a slight skin reaction), purging, cocaine-adrenalin treatment of the mucosa of the nose, ultra-violet light to the sclera; micropsia and metamorphopsia disappeared within six days. The macular oedema disappeared in eight to ten days, leaving deep-lying greyish-yellow spots round the fovea. These did not vanish during the succeeding six years. The colour phenomena faded in one week. It was interesting to note that the order of disappearance of the colours was quite the reverse of their appearance. The red faded last. Four weeks after the beginning of the affection all symptoms had passed away. A relapse after two years in the same eye followed the same course, even the colours coming and going in the same succession as in the first inflammation. But the vision was not so poor as at the first attack (6/12). The greyish spots were to be observed during the whole three weeks. No relapse. Patient examined once a year.

CASE 2.—V.F., aged 37 years; tall, delicate person; old dry pleurisy with incidental increase of temperature. Capillaroscopy —normal findings; no lues; no migraine; no allergic manifestation; blood pressure normal; non-alcoholic; non-smoker.

Left Eye. -3·5 D.Sph. = 6/5.

Right Eye. For the past six to eight days the right eye has been seeing everything smaller and distorted. All objects are coloured dirty greenish. To-day the visual acuity in the right eye = 6/18 with correction.

Right palpebral fissure larger than left (22 : 17 mm.). Movements

up of the right eye are limited; no double vision; retropulsion and extreme movements not painful; anterior part of the eye normal; media clear; disc normal.

In the macular region, occupying the whole area, there is an egg-shaped prominence sharply defined and with an unbroken linear reflex (Fig. 1). The whole prominence is of perfect smoothness, reflecting like glass, without any superficial dots. The extremity only, proximal to the disc, is a little greyish and hazy. The measurements of the prominence are: Horizontal diameter

FIG. 1.

Central serous allergic retinopathy.

4½d.d., vertical diameter 3½d.d. By direct ophthalmoscopy the elevation is 3 dioptres. No pigmentation, no trembling during movements of the eye; no hole in the retina. The whole fovea is brownish-yellow in the light of Vogt's direct current arc lamp. The slope of the lower-outer margin is steeper that than of the upper-inner one. Besides the marginal reflex line there is another reflex arc a little down from the vertex of the prominence, running parallel to the lower margin reflex. The periphery of the fundus is normal. There is no actual central scotoma, but in a central area approximately 10° from the fixation point blue becomes green, white yellowish-green. All other colours are named correctly, but

called dirty. Outer limits of the field of vision normal. Old tuberculin 0·1 mg. intracutaneously, extensive skin reaction, no fever, no local reaction in the retina visible with Gullstrand's binocular ophthalmoscope. Transillumination with Hertzell's diaphanoscope: no shadow.

I could not exclude the possibility of a commencing tumour, but believed it rather to be a local exudate, and treated it with intracutaneous tuberculin, diathermy and diet. For three months

FIG. 2.

Central serous retinopathy—healed.

the prominence became flatter, the micropsia vanished and also the poor vision, as well as the exophthalmos and limitation of eye movement (Fig. 2). After six months all the symptoms had disappeared. Some tiny, deep-lying yellowish-grey pigment spots, similar to snuff-tobacco, in the macular region, remained unchanged (not showing in the photo—Fig. 2). No relapse observed during observation of 1½ years.

CASE 3.—H. (Seen with Dr. I. C. Michaelson). His remarks are: Girl, aged 22 years. X-ray negative. Internal examination normal, slight anaemia. 1936/37: Both eyes inflamed (iridocyclitis).

Left Eye. (June, 1938.) Many corneal precipitates. In the

lower part of the cornea two large ones. Pinkish grey nodules in the ciliary part of the iris.

Right Eye. Posterior synechiae. Lens normal.

Vision : 6/12 right eye; 6/12 left eye, both without correction.

Tuberculin treatment; dionin; U.V. light baths.

Precipitates disappeared within half a year.

May, 1939, eyes white. Vision of the right eye diminished to " fingers " at 4 metres; no micropsia. External appearance normal. Right macular region surrounded by an uninterrupted linear reflex. Slight but definite oval prominence—smooth surface; sharp limit. Treatment discontinued. Patient on holiday.

3.VII. Getting better. Vision = 8/24 after four weeks. Prominence still recognisable. In the centre of the exudate a red spot like a haemorrhage. Periphery normal.

7.VII. 8/18. Exudate within the macular area disappeared.

3.VIII. Vision = 6/9. No macular change.

I think that these three cases certainly belong to the group of *serous central retinopathy*. In all three the changes in the macular region were absolutely evident, in Cases 2 and 3 very similar to each other, with a sub- or intra-retinal transudate (exudate) of oval or circular regular shape. We cannot explain why most of these collections of intra-retinal fluid form a regular circle or oval. That must follow from some physical and anatomical arrangement. As long as effused blood is fluid and sub-hyaloid, it settles with a horizontal upper marginal line according to hydrostatic laws. It is quite different in the case of a fluid gathering in the depth of the retinal tissue where blood extravasates are commonly of circular or oval shape. In the cases mentioned here the scaffolding of the macular region influences the form of these deposits.

It is very difficult to prove an actual spasm of the small macular vessels—Scheerer's endoscopic methods may possibly succeed, but I have no experience of them.

The ophthalmoscopic observation of arteriolar spasm has been recorded several times in the last few years. The authors have usually dealt with young women and with elderly arterio-sclerotic persons. In younger women it occurs suddenly, and constriction of the whole retinal tree or of some branches is observed. The sight is restored after a few minutes. Many of the patients suffer from migraine (H. Friedenwald, 1926). However, no clinical symptoms like ours are known in those cases. The ophthalmoscopic picture of isolated arteriolar branch thrombosis (J. Friedenwald) is a different one.

Further we have to make it clear that our cases did not show signs of Buerger-Winiwarter's endangiitis obliterans or of morbus Raynaud or of quinine intoxication.

The significance of the transient narrowing of the small vessels (capillary spasm) in the ophthalmic investigation of *cerebral* disease is of growing importance. Migraine, for example, is already mostly recognised as caused by a spastic narrowing of certain brain-vessels. The closely related amaurosis partialis fugax with consecutive migraine is often described as the precursor of a so-called embolism—more probably a spastic contraction—of the central retinal artery (Roemer, Löhlein, etc.).

Such a spastic contraction might be of a reflex nature from a focus in the teeth (granuloma), as was proved in a striking case by Redslob (1931). There is no doubt that an arterial constriction in the retina can persist for a prolonged period as this author has succeeded in provoking a spasm of the retinal arteries of some months' duration by one adrenalin injection.

It is very difficult to decide whether the above processes are all based on the spastic narrowing of the vessels only—or if the exudation associated with it influences the clinical picture. No one has proved so far the connection between capillaro-spasm and exudation. The lack of anatomical findings hinders a definite decision.

After a great number of debates in the literature of the whole world, migraine is recognised as an *allergic* disease, with the allergen till now unknown, or certainly not recognised. Allergic *oedema* is emphasised by all observers as the most striking of all allergic processes in the eye tissues, the oedema being due to

FIG. 3.

Allergic bullous dermatitis after applying a strip of plaster at the cheek.

reduction of the colloid osmotic pressure in the vessel-system. We are surprised by the great reaction of the skin after painting the eyelids of an iodine-sensitive person—a reaction which looks like an erysipelas of the most severe kind. Figure 3 shows a 42-year old lady with such a swelling, with large serous vesicles in the skin of the face after the application of a strip of plaster. I have seen a giant oedema of the lids and the whole face half an hour after the patient had enjoyed eating crayfish soup. Intermittent, angio-neurotic paroxysmal oedema of the cornea is described by Foster-Kennedy. It comes from an alimentary source. Schieck, Loewenstein, and others, described the exudative state of irido-cyclitis as an *anaphylactic one* in conformity with the results of many animal experiments. Meniere's vertigo, very often combined with Quincke's oedema and transient iritis is explained by allergic spasm of the vessels. Haziness of the centre of the field of vision has been described by Plumer. There was an oedema in the macular region, with an internal allergy against a special kind of food (chicken meat and walnuts). I saw a so-called embolism of the central retinal artery (better spasm), with a primary retinal oedema in a 47-year old man who had had all his life an intensive allergy against milk.

Klinge's (1930) well-known experiments were dedicated to the clearing up of the aëtiology of rheumatism of the joints and muscles. He injected horse-serum subcutaneously into rabbits and re-injected some of the same serum into the joint after some time. He produced very great changes in the joint, and even very characteristic nodular foci in several spots of the body (myocardium and cardiac valves, tendons, muscles). Swelling, waxy degeneration and necrosis play a great part in producing the anatomical changes. Especially in the endothelium of the small vessels the enlargement of the cells leads to narrowing of the lumen and finally to an actual occlusion of the vessel followed by disturbance of the nutrition of the tissue. Oedema of the mesenchyma is nearly always observed as the first consequence of the vessel blockage by the allergic noxa.

F. Maxwell Lyons (1939) in the eleventh annual report of the Memorial Ophthalmic Laboratory of Giza describes a most remarkable fact about spring catarrh. The conjunctiva dries out in these cases under observation with the slit-lamp, and tiny droplets like dew appear all over the exposed surface of the conjunctiva of the lid. They increase in size until they coalesce and form a thin but tenacious film. The membrane contains a true exudation of fibrin and blood cells which must come from the sub-conjunctival capillaries. In my opinion these findings of Maxwell Lyons leave absolutely no reason to doubt the allergic nature of spring catarrh, sometimes argued against. Indeed the exudation and the

localised chemosis at the limbal region, described as the earliest sign of bulbar involvement by this author, go to prove the allergic character of the affection. Hans Horst Meyer and Chiari published twenty years ago the surprising fact that doses of calcium are able to stop the serous exudation of the nasal and conjunctival mucosa. So it is easily understood that calcium can heal the pathological leakage in spring catarrh by tightening the vessels, and thus improve the clinical manifestations—as Maxwell Lyons has shown in his patients.

All the facts quoted here support the opinion that an allergic vessel reaction is released in cases of allergy, the cause of which often remains unknown, which is followed by a striking oedema.

Very often it is a hereditary structure of the cells which determines the quality of reaction with one or several antigens. That is if the antibody is in the cell plasma pre-existing, and antigen (allergen) brought into association with this antibody release a new substance of histamin character, and this works in an angiospastic manner. The mucosa of the intestine, the tissues of the brain, the conjunctiva and the mucosa of the nose of these patients contain these antibodies. But these antibodies arise too on exposure to certain kinds of infection by organisms of low virulence diffused into the blood from a distant focus. More often the tubercle-bacillus forms a chronic focus with a regular production of allergy. A new batch of the micro-organism from the spreading focus meets the allergic tissue, the combined allergen-antibody frees the toxic substance, and there is a spastic reaction of the vessels which leads to reduced nourishment and oedema. *"All inflammatory features of tuberculosis are allergic"* (A. E. Krause).

Not infrequently we observe in a fresh tuberculous scleritis nothing but a circumscribed chemotic swelling of the conjunctiva. This disappears in the next few days, and then we recognise the infiltration of the sclera. If the scleral infiltration is situated very deeply, exudate moves not only towards the conjunctiva, but also towards the retina. Where it collects—sub-choroidally or sub-retinally—depends on circumstances not known to us exactly (inflammatory adhesions, concentration of albumen, etc.).

I take Case 2 to be a posterior scleritis, with allergic reaction. The inflammatory reaction towards the orbital side causes swelling of the retrobulbar tissues and hence the exophthalmos and the limitation of eye movements, the serous exudation under or in the retina, the prominence in the macular region. There was no exophthalmos in Cases 1 and 3, where the spastic allergic inflammation of the choroid produced an exudate towards the retina only. No actual caseous tubercle developed. The tissue resistance was sufficient to overcome the bacilli present and to render them harmless. It is probable that the previous tuberculin treatment

in Case 3 shared in this favourable biological reaction. The production of exudate in this case had temporarily a worse effect than in Case 1 or 2—the transient reduction of the visual acuity going further.

Case 3 is so far clearer, as here the specific inflammation of the iris with its typical tuberculous nodules does not leave any doubt about the character of the disease.

Czillag (1937) described a case of scleritis with oedema of the conjunctiva in a woman with multiple sclerosis. At the site of the scleritis a nearly total detachment of the retina arose. With X-ray and Ektebin treatment, the scleritis healed simultaneously with the detachment. Visual acuity rose from 1/60 to 5/5. In this case exudate spread in both directions as in our Case 2, producing chemosis of the conjunctiva and detachment of the retina. The retina returned to its old place when the production of exudate stopped, as no cause for the progression of the detachment was present.

Steffen Lund (1938) published a similar case. It was in a woman of 50 years treated for tuberculosis at the Finsen Institute and at the Eye Clinic, with several relapses of scleritis in both eyes, sometimes combined with severe oedema of the lids and chemosis. While suffering from right-sided scleritis, the patient had a relapse in November, 1935, with oedematous swelling of the bulbar conjunctiva, an extensive detachment of the retina in the same eye, both that and the abscess of the sclera which was developing being on the nasal side. The abscess disappeared after treatment for three weeks, the retina became more and more adherent, and finally healed, with isolated radial pigmentations and a visual acuity of 6/6. There seems to be no doubt that the scleritis had given rise to the detachment of the retina, the simultaneous occurrence, the same position, the parallel running healing, not allowing any other explanation. Rumbauer's luetic scleritis (1938) with detachment may be explained on the same lines, but not with the same exactness as the retinal detachment and the scleritis did not run on parallel lines.

Other cases belonging to this interesting group have been published by Purtscher, Kamocki, Pichler. I saw a similar, but progressive case.

F.K., aged 58 years. Fair-haired, easily excited, with skin erythema very often over face and breast. Myopia 6 D.

Vision: Right eye = 6/8; left eye = 6/12.

Fundus: Slight myopic changes.

Left Eye. Conjunctiva of the outer-lower quadrant detached in the shape of a prominent chemotic vesicle, which persisted two months. Excision. Three months after the excision, which healed

easily, patient complained of worse vision, with a curtain from the right side. Almost complete retinal detachment was present, the peripheral part of the inner-upper quadrant only being not detached. Horse-shoe hole very small (¼ P.D.) at 4 o'clock—extreme periphery.

Vision. Fingers at 1½ metres eccentrically. Diathermic occlusion of the hole, with ball and perforating needle. Six weeks afterwards vision = 6/18, right eye = 6/8.

Field of vision normal. Retina in position. Histologically the excised vesicle contained a loose fluid-impregnated connective tissue, without specific cells.

The hole in the detached retina corresponded with the site of the sub-conjunctival episcleral exudate. I believe that we may explain this case by assuming an exudative process in the sclera producing a fluid stream against the conjunctiva as well as against the retina.

As a rule posterior scleritis is not detected, since clinical symptoms are often not present. An anatomical examination has only been made once (by Emanuel, 1902), so far as I am aware. He found a circumscribed tuberculous infiltration of the sclera, with caseation and giant cells, in the eye of a ten year old boy, at the posterior pole of the eye. The choroid was pushed forward at the site of inflammation, and an exudate was detected at this spot between the retina and the choroid. The rest of the retina was adherent round about with extensive scars.

Wagenmann described a case of tuberculous uveitis in a sixteen year old girl with a homogeneous exudate over the part of the choroid not so much changed. A detachment of the retina had developed.

According to our clinical and experimental experience the allergic-inflammatory process reacts by producing an exudate in its beginning only. That is understood from the case of Ginsberg (1910), where three foci of tuberculous uveitis clinically and histologically were found. In the youngest only an exudate was detected, at the site of the two older ones it was already re-absorbed. Detachment of the choroid following a relapsing scleritis is described by Fleischer, and others corresponding to the position of the scleral inflammation. The detachment of the choroid disappeared without residue before the scleritis had gone completely.

Usually the exudate is absorbed, when the inflammatory process of the sclera heals. The detached retina returns to its position, without diminution of its function. The patient in our last case was an older myope—one with the well-known biological inferiority of the retinal periphery. The Iwanoff-Blessig cysts and minor ones are the locus minoris resistentiae, where the fatal rupture sets in. It can be readily understood that the degenerated

myopic retina, lifted by the exudate, may be injured and perforated, while a normal one would resist, and, after re-absorption of the exudate, be re-attached. The occurrence of a peripheral hole is the reason for progression in such a case of exudative detachment of the retina.

The conditions for the occurrence of an exudative retinal detachment are given when:

1. An angiospastic reaction in the sclera or choroid causes an exudate. 2. The exudate finds its way between the choroid and the retina. 3. There are no widespread adhesions between the choroid and the retina. 4. The intra-ocular pressure is not high enough, to press the retina on to the choroid.

A *progressive* retinal separation occurs when there is a tearable spot in a senile or myopically degenerate retina.

Experiments with rabbits (Wessely, 1921, Weekers, 1925, Loewenstein, 1926) have proved the fact that a detachment of the retina can be produced by cauterisation of the sclera. It is very easy to evoke by a slight touch with Wessely' steam cautery. The detached retina replaces itself again in a few days in the majority of cases, but in some the detachment progresses and becomes a total one. We produce here a blister in the interior of the eye raised by burning—an exudate analogous to that which accompanies an allergic inflammation.

Redslob (1931) describes a case of detachment of the retina without a retinal defect, but with a congestion of all choroidal vessels. Circulatory disturbances with physico-chemical variations of the nourishing fluids are assumed to be the cause of the detachment. Histological findings of Mawas (1935) confirm the description of Redslob, and stress the important rôle of the pigment epithelium for the retinal function. Redslob and Nordmann (1938) publish the histological picture of experimental retinal detachment in a dog, produced by a ligature of the vortex veins according to Koster. Extremely choked choroidal vessels with oedema of the pigment epithelial cells are to be seen in the spots corresponding to the detached retina. An upset in the choroidal vessel function is able to provoke a retinal detachment with cystoid degeneration even in a normal eye. Here we ignore intentionally the rôle of pigment epithelium and its discharge (Koyanagi, 1937), and do not mention how far damage of the qualitative and quantitative filtering power of Bruch's glass membrane influences the rise of sub- and intra-retinal exudates. (Loewenstein, 1940.)

The importance of exudative inflammation in causing detachment of the retina has been stressed very often by many authors (Dor, Lauber, Dimmer, Meller, Schall, Loewenstein, Korarskaja and Jorkima, Arruga). All these authors think that a tuberculous or other chronic inflammation of the choroid must not be under-

estimated in the aetiology of retinal detachment. Czillag's, Steffen Lund's and my Case 4 are illustrations of this occurrence. The course of the disorder will be different according to whether the exudate meets a normal or a degenerate retinal tissue, in other words whether it produces a hole or tear in the retina or not. The well-known and not uncommon cases of spontaneous healing of retinal detachment belong mostly to the second group. The cases with retinal defect will be progressive, even if the primary process is healed. A specific treatment in all cases suspected of a tuberculous aetiology should be recommended.

We do not know why in certain cases the exudative fluid oozes into the anterior retinal tissue and in others remains in the deepest layers or sub-retinal. That may depend on the chemical constitution of this exudate. The higher the content of albumen, of fibrin, of cells, or debris, the more difficult will be the penetration of the living retina. The condition of the retina must not be neglected.

There is no doubt that a central angio-neurotic retinopathy does occur. The majority of published cases—it is a small part of those observed, and a far smaller part of those actually occurring —must convince everybody. The lability of the small vessels plays a great rôle in its aetiology.

There will always be the question whether spastic contraction of the retinal vessels may produce the intra- or sub-retinal oedema or exudate. I am not sure about this, and want to stress the point that the choroidal vessel system is at least of the same importance for retinal nourishment, and that far-reaching changes of the ciliary (uveal) vascular system are detected in retinitis albuminurica (Koyanagi and many other authors). The cases described here are in my opinion to be explained as a sequel of choroidal reaction in the first place, even if I do not exclude the effect of spastic contraction of the retinal vessels within the area round the fovea. The retinal pictures published up till now do not seem to be very convincing, but that may be due to nothing but a technical difficulty in the reproduction of such slight changes.

In analogy with migraine we have to emphasise the tendency (usually hereditary) of the capillaries to react to a certain stimulus with spasm, causing disturbance of nutrition within the area of distribution and serous imbibition of the surrounding tissue. The nature of this stimulus is unknown in the majority of cases. The importance of the vegetative nervous system is apparent, and its connection with the endoctrine secretion. Toxic influences are possible, *e.g.*, quinine, alcohol, nicotine, ergotin, mercury, etc. But the significance of allergic capillary spasm is not to be doubted. The commonest infection of an eye tissue is the tuberculous one, which is closely connected with the production of an allergic state of the infected tissue. *Exudation is the essence of*

this allergic response. (A. K. Krause.) Our Cases 2 and 3 belong to the group of retinopathia serosa allergica tuberculosa. The importance of spasm of the small macular vessels of previously unknown origin is stressed by Horniker, Gifford and Marquart, Bailliart, Paterakis and other authors. It is very difficult to say from the long list of published cases how many are allergic. I believe that there is a great number of them. But we cannot prove this assumption. We are much better informed about the allergic origin of the Cases 2 and 3, especially in Case 3, with the typical tuberculous changes in the iris tissue.

So I come to the conclusion that the deep cleavage between the opinions of Horniker and Gifford on the one hand and Kitahara and Riehm on the other, is more fictitious than it seems to be on first impression. It is just possible that we may be able in a short time to *prove* many of the central angio-spastic retinopathies to be allergic.

Summary

One case of a central retinal process is understood to be an angio-neurotic retinopathy of unknown, probably allergic, origin. A second case with exophthalmos and limitation of eye movements with sub- or intra-macular exudate is diagnosed as posterior scleritis with exudation both into the orbital tissues and sub-retinally. A third case shows a central fundus picture analogous to the second one in connection with a typical tuberculous iridocyclitis. A fourth shows a chemotic sub-conjunctival bleb with, later on, corresponding retinal detachment in a myopic eye. The author emphasises the fact that the allergic inflammatory reaction—tuberculous or non-tuberculous—is always characterised by a primary narrowing of the smallest vessels with an oedematous imbibition of the surrounding tissue. The allergic spasm of the retinal and choroidal vessels plays a dominant part in the development of the central retinopathy. Specific processes in the choroid and sclera with exudation cause retinal detachment. In the myopic or senile retina, the occurrence of exudate in the presence of cystic degeneration may easily lead to the production of a tear in the retina and a detachment of progressive character.

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A CASE OF BLUE SCLEROTICS

BY

J. P. SPENCER WALKER

HOVE

THIS case of blue sclerotics may be of interest to your readers. The association of deafness and liability to fracture of bones is well known, but there is some doubt as to whether the sclera is thin and so the choroid shows through, or is of normal thickness but transparent.

There was a history of deafness of the left ear of long standing, and of recent fracture of the femur on stepping off an omnibus. As far as she knows, none of her relatives is deaf, has blue sclerotics, or has sustained fractures. The patient was brought to me by her doctor, as she had almost lost the vision of the left eye. On examination the blue sclerotics in both eyes were very noticeable, the vision of the right eye was 6/6, and of the left perception of light only. The tension of the right eye was normal, but that of the left was raised considerably. The field of vision of the right eye was normal. The patient is aged 46 years.

As the tension did not lessen with the use of miotics, I decided to trephine the left eye. Though I expected the sclera might be thin, I was surprised to find how very thin it proved to be. One complete turn of the Elliot's trephine was sufficient to cut through.

My object in reporting this case is not to discuss the treatment of the glaucoma, but to draw attention to the thinness of the sclera, and to the absence of stretching of the sclera with such an increased tension in an eye with so thin an outer coat.

TUBERCULOMA OF THE EYE

BY

G. E. DODDS, M.B.

WEST AFRICAN MEDICAL SERVICE

IN this case the disease had reached a stage which would rarely be seen in Europe to-day and for this reason may prove of interest. Very little history could be obtained—the patient, a male Yoruba, aged about 12 years was brought to the eye clinic at Lagos with the condition as seen in Fig. 1. He also had advanced spinal caries

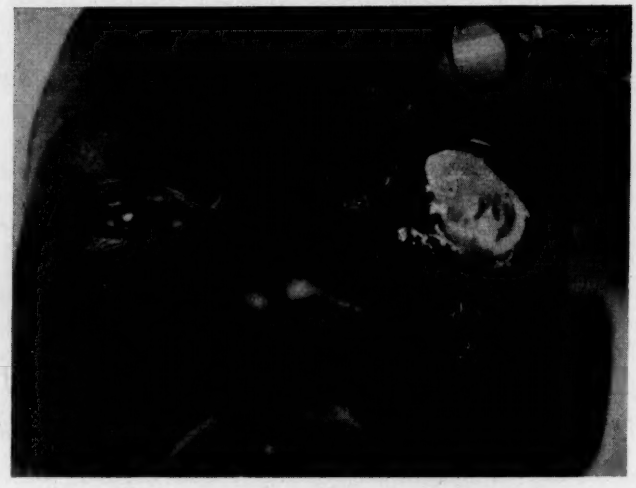

FIG. 1.

with spastic paralysis of the lower extremities. He was admitted to the hospital and treated by extension and tonics.

His condition was considered hopeless and it was thought that an early death would put an end to his troubles. His mother stated that about two years previously he complained of pain in the back and later that a swelling had developed on the back; his legs then became powerless and finally the eye trouble started. No other details of the course or the appearance of the disease could be obtained. After three months treatment the patient's general condition had improved and the tumour had diminished in size—see Fig. 2—photograph taken three months after Fig. 1.

The globe was enucleated and the pathologist, Dr. D. A. Cannon reported as follows:—

FIG. 2.

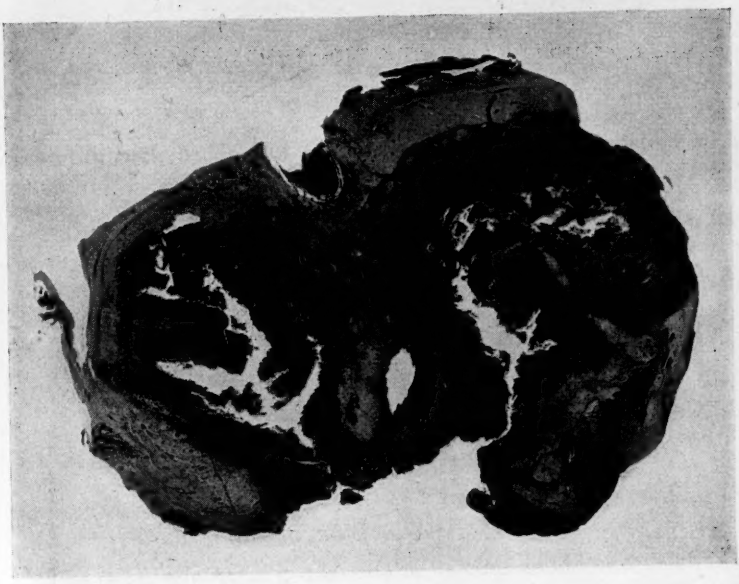

FIG. 3.

The enucleated eye consisted of a soft, solid mass which bore little or no resemblance to an eye-ball; the remnant of the lens and cornea were still visible, but for the rest, the organ consisted of a mass of necrotic tissue, bathed in creamy, yellow pus; no cavity of the eye persisted. Sections revealed merely a mass of caseous tissue, in which were numerous typical large tuberculous giant-cells,

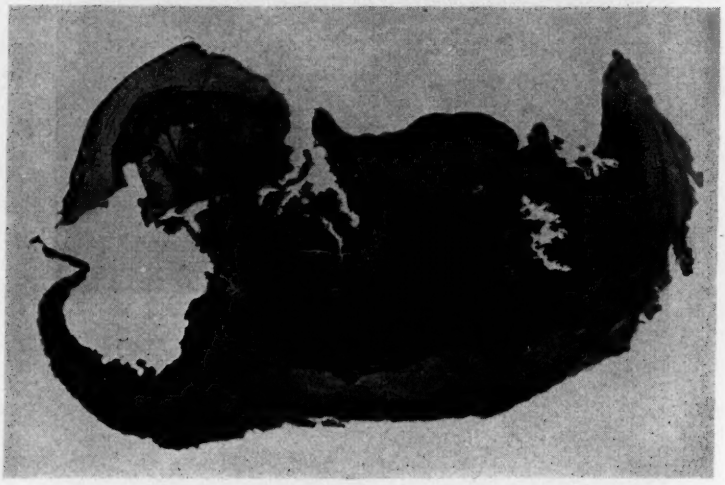

FIG. 4.

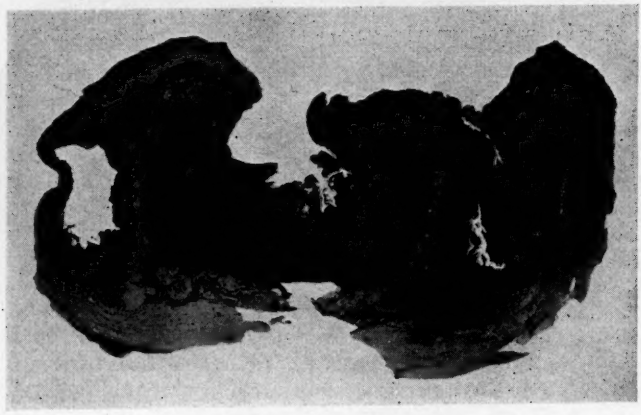

FIG. 5.

the process of caseation had involved all tissues of the organ (see Fig. 3).

Fig. 4 shows a part of the ciliary body, which is broken up and largely destroyed; small foci of caseation, with giant-cells can be seen to be invading the several layers of the sclera (see Fig. 6, H.P. of Fig. 4). Direct smears made from the enucleated eye failed to

FIG. 6.

reveal tubercle bacilli, but a guinea-pig, which was inoculated with pus from the eye, was killed after seven weeks, and showed typical tuberculous lesions which contained acid fast bacilli.

I wish to thank the Director of Medical Services, Nigeria, for permission to publish these notes; Mr. Knight of the Nigerian Laboratory Service for photographs and the Pathologist, Dr. D. A. Cannon.

ANNOTATIONS

"WHAT'S IN A NAME?"

"What is your Name?" "N or M."

"Who gave you this Name." "My Godfathers and Godmothers in my Baptism." *The Catechism*

The responsibility of godparents in naming children is great. It is clearly a risky procedure to call a girl "Prudence" or

"Constance"; and the puritanical habit of inflicting such Hebrew designations as Isaac, Jabez, or Kerenhappuk is liable to arouse unchristian reactions in the victims. It is no doubt a wise precaution which makes it difficult for a person to change his name, and it might be a great advantage if the re-christening of diseases and medical terms could be effected only by deed-poll administered by a properly constituted legal body, *e.g.*, a reformed General Medical Council, making it a criminal offence to change the name without overwhelming proof of the superiority of the new one.

A name is a symbol, and the first requisite is that it should be generally intelligible. In this respect the Catechism falls short of perfection, for "N or M" is, to say the least of it, ambiguous. "N" certainly suggests "nomen" to the intelligent antiquarian; but what of "M"? It requires antiquarian research to discover that "M" stands for "NN," an abbreviation for the plural of "nomen," and that it has arisen from the mistaken reading of almost illegible manuscripts.

It is difficult to find a good criterion for the nomenclature of diseases, and it is indeed impossible in the present state of knowledge. It is generally agreed that the most scientific is the pathology of the disease, when this is known. Unfortunately pathologists do not always agree, and they are no less susceptible to the allurements of novae res than other people. For example, we have long been accustomed to speak of malignant tumours of the uveal tract as sarcomata, on the assumption that they are derived from mesodermic cells. On the basis of some exceedingly interesting and suggestive work many—apparently most—pathologists have come to the rather surprising conclusion that they are derived from the cells of the sheaths of Schwann of the ciliary nerves. It seems to have been more or less universally decided on these grounds henceforth to call these tumours "malignant melanomata." Is this really desirable? In the first place, if we *are* going to change the name it ought to be for a really comprehensive one. Some of these growths are unpigmented, so that "melanomata" does not seem to be an ideal term. In the second place, what is the pathological foundation for the new theory? It is chiefly histological, and is open to other interpretations. It is a great pity that anatomists, physiologists, and pathologists seem often to forget that a stained microscopical section bears about the same relation to the living tissue as an embalmed and painted mummy does to the living body. On the same basis the cells of gliomata of the brain have received a large number of tabloid descriptions in the form of newly coined terms of Greek origin. Is not this sheer nominalism, with all its insidious dangers and no real increase of knowledge? And are we not liable to think that we know all about the cause of retinitis

pigmentosa when we refer it to abiotrophy, which etymologically seems to have something to do with the nourishment of the dead.

Some well established names are undoubtedly bad; but we know what they mean—which is the most important function of a name—and until they can be replaced by others of proven validity and manifest advantage they are best retained. One such is "glioma" of the retina, which differs toto caelo from glioma of the central nervous system, and which a few years ago most pathologists would have preferred to call "neuroepithelioma." Nowadays they want to call it neuro- or retino-blastoma, or retino-cytoma. The crucial question is whether the game is worth the candle. It is scarcely necessary to point out that pharmacologists and drug manufacturers must share with pathologists the opprobrium of multinominalism, but they have the excuse that the authentic names given to synthetic drugs by the organic chemists run to polysyllabic excess.

There is a certain fascination about concocting new terms, and not much harm is done so long as the term is not regarded as embodying a scientific explanation. It is rather like devising new instruments—"many are called, but few are chosen."

Unnecessary Injuries

Once again the National Society for the Prevention of Blindness has called attention to the great number of needless injuries caused by fireworks, especially among children, in the Fourth of July celebrations. Last year these celebrations were the cause of 214 serious eye accidents, 15 of which resulted in blindness in one or both eyes. The total number of fireworks injuries of all kinds on July 4, 1940 was reported by the American Medical Association as 4,462. New York State headed the list with 1,114, and of these 32 were major eye accidents, four of which resulted in loss of vision of one or both eyes.

Indiana, on the other hand, showed a very great improvement: there were only two injuries reported in 1940, as against 198 in 1939. The State's law against fireworks became effective for the first time last year.

This is not the first time that we have commented on the subject, and at the present time when this country is being subjected to nightly attacks by bombs and fire, we can all sympathise with the National Society for the Prevention of Blindness in their campaign against needless injuries.

ABSTRACTS

MISCELLANEOUS

(1) **Beetham, William P. (Boston).—Atopic cataracts.** *Arch. of Ophthal.*, July, 1940.

(1) Atopy is a word coined by Coca to describe the occurrence of eczema, hay fever, and lichen of the skin in young persons with a constitutional predisposition to hypersensitivity. The basis of this predisposition is thought to be the presence in the blood or serum of the so-called Prausnitz-Kustner bodies which convey a specific, urticarial sensitivity to normal skin. It is logical to suppose that the lens, being an ectodermal structure, might partake in the general reaction of hypersensitivity, and various authorities have reported cases of this disease in which cataract was an associated lesion; **Beetham**, in his paper, giving details of ten more.

The rôle of allergy in producing the cataract is not, however, certain, because in 30-50 per cent. of cases the involvement is unilateral and it is difficult to imagine that one lens becomes sensitised while the other does not. Also, negative skin reactions were obtained when aqueous from an affected eye was injected intradermally. One feature, common to the cases was malnutrition, the weight of the patients being from 10-25 per cent. below the normal for their age. This peculiarity was unexplained, at any rate so far as dietetic and endocrine factors were concerned. Two distinct types of cataract were found. The first usually began at the posterior pole, with increased iridescence, vacuoles and precipitates, the changes spreading peripherally and then appearing at the anterior pole. Eventually, the entire cortex became cloudy. Less commonly, a dense irregular plaque formed in the pupillary area of the subcapsular cortex with a varying degree of involvement of the remaining lens matter.

F. A. W-N.

(2) **Chandler, Paul A. (Boston).—Intradiem-tension studies in chronic simple glaucoma.** *Arch. of Ophthal.*, July, 1940.

(2) It has long been known that within the same day there may be marked variations of the ocular tension in cases of glaucoma, and **Chandler** decided to investigate these changes in a series of 22 patients in whom the indications for operation were in doubt. The usual plan was to admit the patient to hospital and take readings of the tension at 8 a.m., 12 noon, 6 p.m. and 10 p.m. In a few cases, readings were taken in the middle of the night as well. After a day or two without treatment, miotics were employed and the readings

continued for a further few days. The cases tended to fall into 3 groups though the divisions were not always clear cut.

Group 1 comprised 7 patients in whom the tension was always within normal limits and little, if at all, affected by miotics. In spite of this, disc and field changes, characteristic of glaucoma were present in all. Three explanations are offered—(*a*) That the normal tension for such eyes is in the region of 8-10 mm. Hg and that a tension of 18-25 mm. Hg represents, for them, a glaucomatous state. Proof of this was forthcoming in 3 cases in which miotics were ineffective, but operation which reduced the tension to 12mm. or less prevented further deterioration of fields. In one of these cases, four years after operation, the fistula became partially closed and the tension went up to 20 with a resultant further loss of field. (*b*) The normal tension is 18-25, but there may be periodic unobserved rises. One case seemed to be of this type. She had been under observation for nine years, during which time the tension had remained at 22, except on one occasion when it rose to 35. (c) There has been a glaucomatous state in the past, which has become spontaneously arrested. Three cases of this type were encountered in which, in spite of the presence of glaucomatous stigmata, no further loss of field occurred during the period of observation.

Group 2 comprised 9 cases in which the tension was sometimes elevated but was maintained at a normal level by miotics. One of these cases was observed for as long as six years without showing an additional loss of field.

Group 3 comprised the remainder of the cases. In these the tension was sometimes elevated and was not satisfactorily controlled by any miotic, so that operation was imperatively indicated.

F. A. W-N.

(3) **Koch, C. and Schreiber, G. (Trieste).—Metaplastic bone formation in transplantation of the ureter into the anterior chamber of the eye of the guinea pig. (Metaplastische Knochenbildung bei Transplantation von Ureter in die Vorderkammer des Meerschweinchenauges).** *Ophthalmologica*, Vol. XCVII, p. 284, 1939.

(3) **Koch** and **Schreiber** report experiments in which urethral segments of embryonal and adult guinea pigs were introduced into the anterior chamber of the eye of the guinea pig. In all, eight experiments were made and histologically examined and in each the formation of bone was observed within the newly formed connective tissue of the neighbouring regions of the implant. The formation of bone was plainly to be seen on the 18th day after transplantation. Attention is drawn to the special, if not specific, ability of the urethra to induce the formation of bone.

ARNOLD SORSBY.

(4) **Sveinsson, K. (Reykjavik).—Choroiditis areata.** *Acta Ophthalmologica*, Vol. XVII, p. 73, 1939.

(4) **Sveinsson** describes four cases of extensive peripapillary choroidal atrophy labelling the condition "choroiditis areata." Two of the four patients were a mother and her son, aged 4 years. He points out that islands of healthy tissue may be seen within the atrophic area.

It is questionable whether this is to be regarded as a distinct entity. These cases may represent early stages of gyrate atrophy of the retina and choroid.

ARNOLD SORSBY.

(5) **Persichetti (Rome).—The behaviour of the eye towards intravenous injection of animal charcoal. (Ricerche sperimentali sul comportamento dell'occhio di fronte alle iniezioni endovenose di carbone animale).** *Boll. d'Ocul.*, May, 1939.

(5) The value of animal charcoal as an absorbent antiseptic has been known for a long time. It has been used in the treatment of many acute local and general diseases with profit. **Persichetti** wished to see what happened in the eye after these injections; it seems that no account of this organ had been given previously. He concludes that it is possible to find the carbon in the eye after such injection, but it is generally necessary to repeat the injection more than once. The deposits are found in the vessels and never in the tissues. No pathological change is found in the surrounding tissues and no function is disturbed. There is no evidence that inflammation attracts the deposit of carbon. It is eliminated very slowly, if at all; months after the injection the deposits in the vessels seem unaltered.

HAROLD GRIMSDALE.

(6) **Cordero (Pisa).—Congenital hole at the macula, and persistent ductus arteriosus. (Foro maculare congenitoe persistenza del dotto di Botallo in soggetto con status dysraphicus vasalis).** *Arch. di Ottal.*, March, 1939.

(6) **Cordero** reports a case of much interest. It is that of a girl aged 19 years, whose sight had never been very good; she had suffered from headache and pain in the cardiac region for some time, with other general symptoms. Her corrected vision was R. 7/10 partly, L. 6/10. Ophthalmoscopic examination showed a deep red patch about half a disc diameter in width and rather smaller in height in both eyes. This was separated from the surrounding retina by a narrow whitish halo which faded off all round. There was about 1 D. difference in level between the surrounding retina and the "hole." The visual field showed an absolute ring scotoma about 20° from fixation. The luminous sense was diminished in

both eyes. The arterial pressure was found to be higher on the right side than on the left both in the retinal arteries and in the brachials. From this it was concluded that the ductus arteriosus was still patent. Two figures show the "holes" and the slight difference in colour of the discs.

HAROLD GRIMSDALE.

(7) **Fallica (Florence).—The estimation of volatile aromatic bodies in the rabbit's vitreous. (La titolazione delle sostanze aromatiche volatili nel vitreo di coniglio).** *Boll. d'Ocul.*, January, 1939.

(7) The importance of these bodies is clear when we consider the frequency with which increase of their amount is accompanied by ocular change. It is advisable to know whether the vitreous contains such substances and in what amount. For this estimation, **Fallica** has used the method of Nassi; this depends on the transformation of phenol into tribromophenol by the addition of a solution of bromobromate of potassium. The amount of bromine left after the reaction is then measured.

The author finds the amount of volatile phenols in the vitreous to be somewhat less than in serum.

HAROLD GRIMSDALE.

(8) **Rubino (Padua).—An unusual congenital opacity of the lens. (Su una particolare opacità congenita del cristallino).** *Boll. d'Ocul.*, January, 1939.

(8) **Rubino** describes the condition of the lens in a boy aged 14 years, whose right eye had been accidently found to be defective. The anterior part of the lens was transparent but in the layer behind the embryonic nucleus there was an opacity consisting of a network of spaces filled with punctiform dots.

HAROLD GRIMSDALE.

(9) **Santoni (Naples).—Research on the structure and the metabolism of the retina of the frog during regeneration. (Ricerche sulla struttura e sul metabolismo della retina di rana durante il processo di regenerazione).** *Boll. d'Ocul.*, January, 1939.

(9) It is known that the power of regenerating limbs and organs decreases as animals rise in the scale of complexity; it is less in the vertbrata than in other orders and is limited to the imperfectly developed individuals. In mammals it is limited to the healing of wounds. In larval amphibia even the eyes may be regenerated. **Santoni** has examined the power of regeneration of the retina in the adult frog after division of the optic nerve. He found that within 48 hours degeneration of the retina began; in a

few weeks all the retinal structures had broken down and disappeared. The first signs of regeneration appeared after some three months; the new retina seemed to spring from the region of the ciliary processes, but there were small zones of regeneration in other regions which were due to remaining pigmented epithelium or perhaps to cells migrating from the ciliary processes. In three or four weeks the whole eye was lined by a membrane of several layers, the outermost of which was made up of rods and cones. The metabolism of the retina during the process of regeneration shows a slight increase of anaerobic glycolysis; there is no appearance of aerobic glycolysis.

HAROLD GRIMSDALE.

CORRESPONDENCE

TREATMENT OF MUSTARD GAS LESIONS OF THE EYE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS—An instruction E.M.S.I. 252 (revised) on this subject has been widely distributed to the officers, consultants and hospitals of the Emergency Medical Service. It came before the Medical Board of Moorfields Eye Hospital 'for your information.' The Medical Board considered the instruction and felt that several points in it required comment.

1. The use of albucid solution is advised at First Aid Posts and at Hospitals in cases in which the eyes have been affected by gas vapour or gas splashing. It must be pointed out that the use of sulphanilamide preparations is not directed against gas contamination but only against subsequent infection of the conjunctiva and that opinion as to its efficiency in this latter respect is far from being generally favourable.

2. No mention is made in the instruction of the very diverse lesions which may be produced by gas, most of them slight and very few of them serious.

3. No indication is given of the treatment necessary when the cornea is definitely involved and while water is advised for irrigation of the eye at an incident and at First Aid Posts, no advice is given as to the lotion to be used for the irrigations recommended at Hospitals.

It would appear that this instruction is not in fact the 'result of further experience' and should be withdrawn and replaced by a

carefully considered and detailed note based on actual experience obtained in the last war and on substantiated experimental evidence.

We are,

Yours faithfully,

F. A. JULER.
MAURICE WHITING.

ROYAL LONDON OPHTHALMIC HOSPITAL,
June 16, 1941.

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS—In your issue for March you print a notice of the Thirteenth Annual Report of the Memorial Ophthalmic Laboratory, Giza, in which your reviewer writes that "It is improbable that any ophthalmic laboratory in the World can have had the opportunity of examining 74 malignant tumours of the eye and its adnexa in a single year."

It might be of interest to record the figures for such cases at the Pathological Laboratory at Moorfields in recent years.

Last year, in which the total turnover was the lowest for many years, the figure was 77; for 1938 and 1939 the figures were 87 and 103 respectively.

I am, your obedient Servant,

FRANK W. LAW.

ROYAL LONDON OPHTHALMIC HOSPITAL,
June 16, 1941.

NOTES

Royal Society of Medicine, Section of Ophthalmology

The following officers have been nominated for the session 1941-42. President, A. J. Ballantyne; Immediate Past President, M. L. Hepburn; Vice-Presidents, A. E. Iles, A. F. MacCallan, J. Cole Marshall, O. Gayer Morgan, W. C. Souter, M. H. Whiting; Hon. Secretaries, C. D. Shapland, Harold Ridley; Members of Council: Physicians, D. H. Brinton, D. Denny-Brown, S. P. Meadows. Metropolitan Members, E. F. King, Mary A. Pugh, F. Ridley, R. S. Scott, R. E. Bickerton, V. B. Purvis, A. J. B. Goldsmith, P. M. Moffatt, H. H. Skeoch. Provincial Members: G. T. Willoughby Cashell, E. G. Recordon, G. W. Black, N. Cridland, J. G. D. Currie, G. Ian Scott; Representative on Library Committee (Hon. Librarian of the Bowman Library), Arnold Sorsby; Representative on Editorial Committee, E. Wolff.

White Oak Hospital, Swanley. The following are the admissions recorded for 1940.

Phlyctenular Ophthalmia	44
Blepharitis	42
Trachoma	5
Interstitial Keratitis	3
Acute Conjunctivitis	21
Chronic Conjunctivitis	10
Miscellaneous...	16
	141

In addition 131 infants and 93 nursing mothers were admitted to the Ophthalmia Neonatorum block (transferred as a war measure from St. Margaret's Hospital.)

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Sight Conservation. The third number of the 6th volume of "Neighbourhood Health" is devoted to sight conservation. It is a well produced pamphlet of 12 quarto pages and contains interesting articles and good illustrations. Among the contents we note a paper by Rice, on the Health Department's rôle in the prevention of blindness, one by Berens, on advances in eye surgery, an excellent paper on the story of eye glasses, and shorter notes on preventing blindness, by Miss Merrill, on syphilis of the eye, by Kirby, and gonococcal infections of the eye by Thygeson.

The eyes in advancing years by Hardy, and eye protection in industry by Greenburg, also call for favourable comment. A short column of questions and answers, entitled "Do you Know" is excellent and the illustrations and general set-up of the whole are admirable.

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D.O.M. and S. Eng. THE following have been elected examiners for the D.O.M.S. for the ensuing year by the Royal College of Surgeons of England. Part 1, Mr. Frank Law and Mr. L. H. Savin. Part 2, Mr. Adrian Caddy.

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The Tennent Chair of Ophthalmology, Glasgow PROFESSOR A. J. BALLANTYNE, the first holder of the Tennent Chair of Ophthalmology in the University of Glasgow, retires this year under the age limit. As his successor, Mr. W. J. B. Riddell has been elected as from October 1, 1941.

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Leslie Dana Gold Medal, 1941 THE Leslie Dana Gold Medal for outstanding achievements in the prevention of blindness and the conservation of vision for the year 1941 has been awarded to Dr. Arnold Knapp, of New York.

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Corrigendum WING COMMANDER T. KEITH LYLE draws our attention to a slip in proof correcting on page 295 of the June No. In paragraph 5 of the Conclusions ("Dosage") 250 mgms. should read 50 mgms.